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REVIEW

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Radiological assessment of hydrocephalus: new theories and implications for therapy

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Abstract It is almost a century since Dandy made the first experimental studies on hydrocephalus, but its underlying mechanism has been unknown up to now. The conventional view is that cerebrospinal fluid (CSF) malabsorption due to hindrance of the CSF circulation causes either obstructive or communicating hydrocephalus. Analyses of the intracranial hydrodynamics related to the pulse pressure show that this is an over-simplification. The new hydrodynamic concept presented here divides hydrocephalus into two main groups, acute hydrocephalus and chronic hydrocephalus. It is still accepted that acute hydrocephalus is caused by an intraventricular CSF obstruction, in accordance with the conventional view. Chronic hydrocephalus consists of two subtypes, communicating hydrocephalus and chronic obstructive hydrocephalus. The associated malabsorption of CSF is not involved as a causative factor in chronic hydrocephalus. Instead, it is suggested that increased pulse pressure in the brain capillaries maintains the ventricular enlargement in chronic hydrocephalus. Chronic hydrocephalus is due to decreased intracranial compliance, causing restricted arterial pulsations and increased capillary pulsations. The terms "restricted arterial pulsation hydrocephalus" or "increased capillary pulsation hydrocephalus" can be used to stress the hydrodynamic origin of both types of chronic hydrocephalus. The new hydrodynamic theories explain why third ventriculostomy may cure patients with communicating hydrocephalus, a treatment incompatible with the conventional view.

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Introduction

The conventional view on the cerebrospinal fluid (CSF) circulation is that of a bulk flow, from the site of production at the choroid plexus to the site of absorption at the pacchionian granulations (Fig. 1). The driving force of bulk flow is the CSF pressure at the production site being slightly in excess of the pressure at the absorption site (Fig. 2). The CSF bulk flow theory explains hydrocephalus as an imbalance between CSF formation and absorption. An obstruction to the CSF flow, inside or outside the ventricular system, causes obstructive and communicating hydrocephalus, respectively. The intracranial pressure is thought to be dependent on the balance between production and absorption of CSF. This indicates that patients with hydrocephalus should have increased intracranial pressure. The bulk flow theory makes the pathophysiology of hydrocephalus both easy to understand and possible to summarize briefly. However, the theory has proven incorrect, mainly because the major absorption of CSF occurs in the capillaries of the central nervous system and not in the pacchionian granulations.

The application of the bulk flow theory on hydrocephalus originates from Dandy [1], who made the first experimental studies on hydrocephalus in 1914. He plugged the aqueduct in dogs and found that the ventricles dilated proximal to the obstruction. This is the first evidence that CSF is produced in the ventricles and that the intraventricular absorption of CSF is less than this production. Although Dandy proved that an intraventricular CSF obstruction causes obstructive hydrocephalus, he recognized that decreased bulk flow across the pacchionian granulations could not cause communicating hydrocephalus. He questioned that the pacchionian granulations

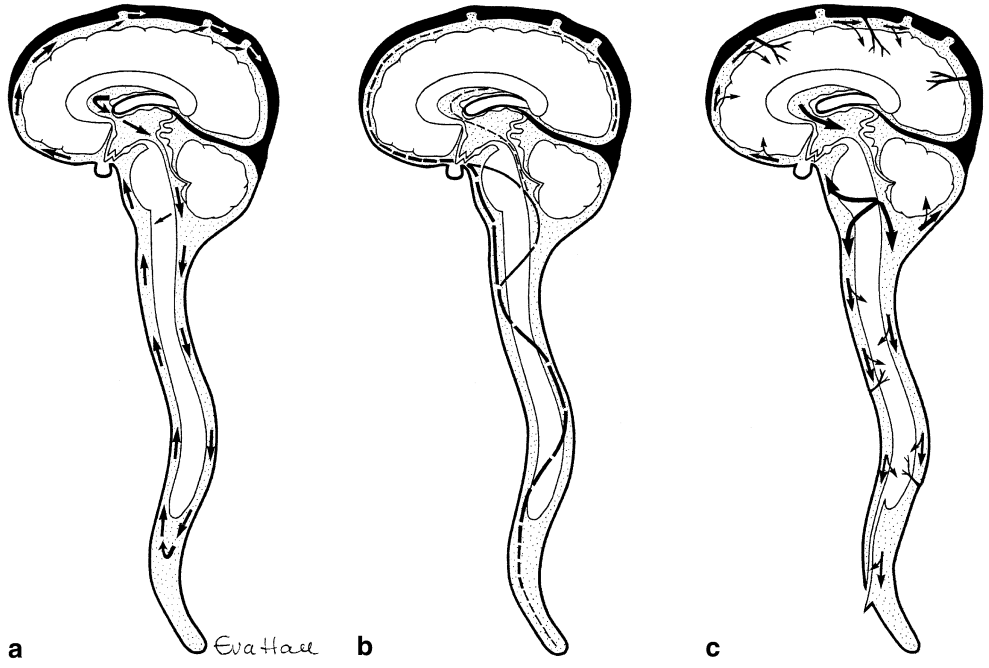

Fig. 1 Diagram showing the **a** bulk flow model and **b**, **c** the two types of CSF circulation related to the revised concept of circulation. **a** CSF is produced in the choroid plexus and is transported by bulk flow to the arachnoid granulations at the venous sinuses, where it is absorbed by a valvular mechanism. **b** There is a dominant pulsatile CSF flow, which is responsible for the transport of CSF.The transport of CSF occurs by mixing caused by intracranial arterial pulsations. The length of the segments of the *dashed line* indicates the magnitude of CSF velocity. There is a fast CSF velocity and transport compartment in the brain stem–cord area and slow CSF velocity and transport compartments in the upper and lower ends of the subarachnoid space. The caudal systolic and the cranial diastolic CSF flows in the spinal canal follow one main channel along the spinal convexities. **c** The minute CSF bulk flows are exaggerated for more clear illustration. The thickness of the *arrows* is related to the magnitude of bulk flow, which decreases in both directions from foramen magnum. The CSF is absorbed everywhere in the central nervous system by the capillaries of the central nervous system (permission granted from Am J Neuroradiol 1996;17:431–438)

absorb CSF and stated that the ventricles should not dilate, if there is a CSF blockage at the pacchionian granulations. Such an obstruction cannot cause a higher pressure in the ventricle than in the subarachnoid space, but would instead dilate the subarachnoid space (Fig. 2d). By injecting a colour tracer intrathecally and measuring its excretion in urine, Dandy provided compelling evidence that the CSF absorption is a diffuse process occurring everywhere in the capillaries of the subarachnoid space [1]. Since the CSF absorption capacity of the cranial subarachnoid space exceeds the CSF production, he excluded idiopathic communicating hydrocephalus from the bulk flow theory and left this condition unexplained.

Later, O'Connel [2] and Hakim [3] described patients with the clinical syndrome of idiopathic normal-pressure hydrocephalus (NPH), i.e. the triad of mental deterioration, gait disturbance and urinary incontinence associated with communicating hydrocephalus and normal ventricular pressure. NPH is also incompatible with the bulk flow theory [4, 5]. The ventricles should not dilate without an increase of the CSF pressure, and the CSF pressure in turn should increase at CSF outflow obstructions. Greitz [6–10], using flow-sensitive magnetic resonance imaging (MRI) and radionuclide cisternography, rediscovered that the brain capillaries absorb CSF (Fig. 1b,c). Other researchers using different CSF tracers have verified this [11]. This revised view on the CSF circulation prompts a new explanation for the development of communicating hydrocephalus. In 1943, O'Connel for the first time correctly postulated that increased CSF pulse pressure in the ventricles, without increase of mean CSF pressure, could cause communicating hydrocephalus [2]. Consequently, modern theories emphasize the importance of vascular pulsations and vascular absorption of CSF for the pathophysiology of communicating hydrocephalus.

Normal CSF physiology and brain water

Critique of the CSF bulk flow theory

The evidence for the bulk flow theory (Fig. 1a) [12] came in the 1960s, when Welch and Friedman reported that the pacchionian granulations could act as mechanical valves [13], and Di Chiro found that a late, 24-h maximum of radioisotope accumulation was seen around the pacchionian granulations at radionuclide cisternography [14]. However, it is difficult to reconcile the bulk flow theory with other observations: (1) the pacchionian granulations do not develop in children until the closure of the fontanels, (2) no mechanical valves have been demonstrated anatomically in the pacchionian granulations, (3) radioactively labelled albumin appears in blood within minutes after injection into the lumbar CSF, (4) 80–90% of

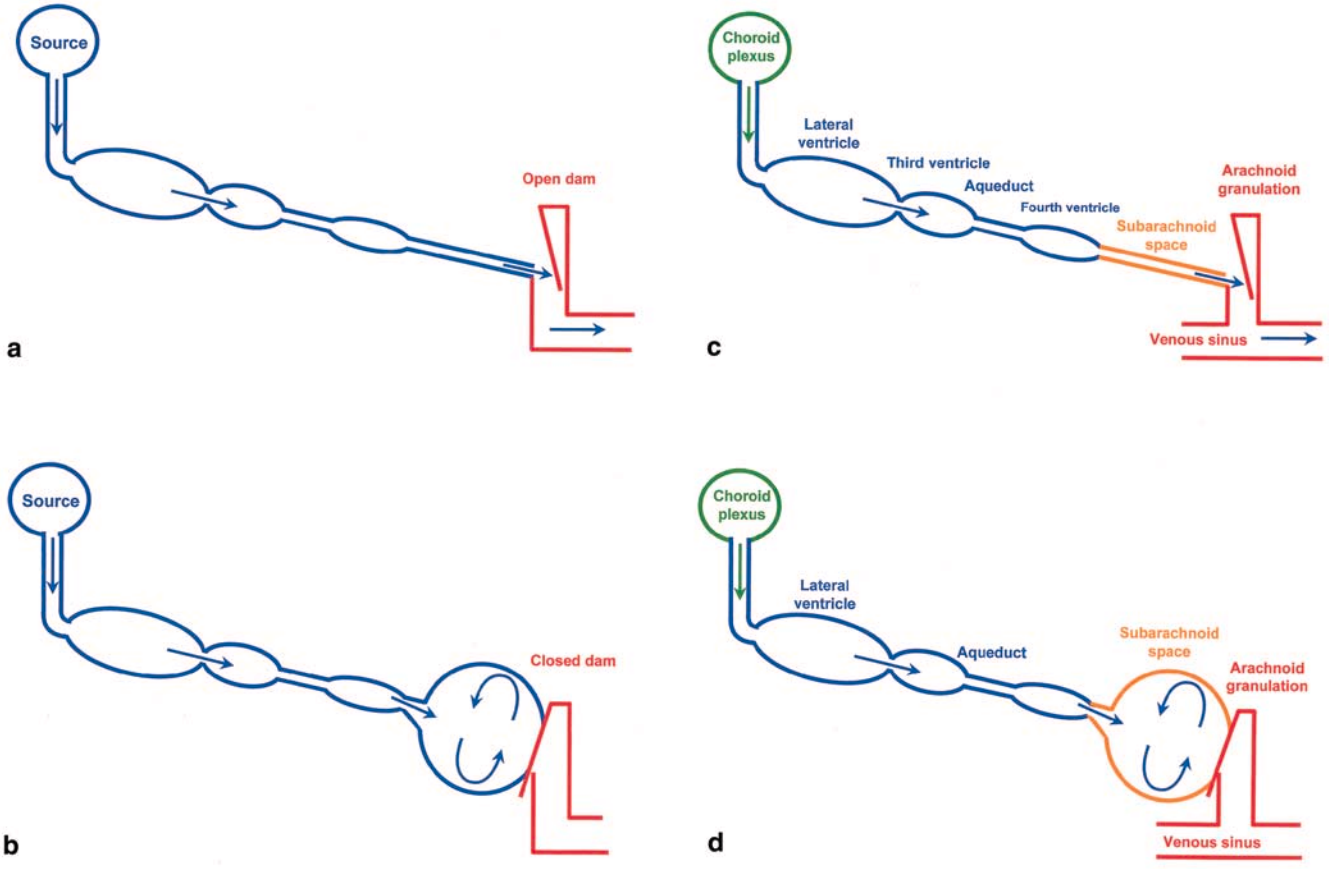

Fig. 2 **a**, **b** Schematic drawing of a river with interconnected lakes and outflow through a dam. There is a pressure drop from the source to the dam. When the dam is closed, there is an enlargement of the fluid space just upstream to the obstruction (**b**). **c**, **d** The bulk flow concept. The driving force of the CSF circulation is a pressure drop from the choroid plexus to the arachnoid granulation. As is the case in the river, the subarachnoid space, not the ventricular system, would dilate when the outflow through the arachnoid granulation is closed (**d**)

the injected radioisotope is absorbed in the spinal canal, and (5) a late maximum of the radioisotope is present also in the lumbosacral area [6, 8–10]. In fact, the 24-h isotope maximum at the convexity and lumbosacral area indicate regions with decreased turnover of CSF. This means that the classical interpretation of radionuclide cisternography, that the CSF tracer in the subarachnoid space is transported by bulk flow, is incorrect [8].

Until now, no experimental evidence or quantitative measurement exists, which proves that fluid is transported across the pacchionian granulation. This contrasts to the well-documented fluid exchange across the wall of the brain capillaries. It remains a riddle why the fluid absorption by brain capillaries has not been considered, when discussing the CSF absorption mechanisms. Starling [15] discovered the fundamental principles behind fluid homeostasis already in 1896. The Starling principle states that two counteracting forces within the capillary balance the fluid exchange across the capillary wall, i.e. the hydrostatic pressure causing fluid filtration and the colloid osmotic pressure causing fluid absorption. It has been accepted as a general mechanism for fluid homeostasis in the body for almost a century. It is mandatory to apply the Starling principle also on the absorption of interstitial fluid and CSF in the central nervous system, where the crystalloid osmotic pressure in the brain capillary determines the fluid absorption [16]. As mentioned, Dandy found compelling evidence that the absorption of CSF is a diffuse process occurring everywhere in the subarachnoid space. Greitz confirmed that brain capillaries absorb the CSF and that CSF is transported by vascular pulsations in the subarachnoid space [6–9, 17]. He also postulated rapid and unidirectional transport of proteins and macromolecules by endothelial vesicles in the opposite direction of the blood–brain barrier, from the brain tissue to blood [6, 9].

Modern CSF physiology and brain water

CSF is produced everywhere in the central nervous system. The main production occurs in the choroid plexus, and the absorption of CSF occurs in the capillaries of the central nervous system (Fig. 1c). The rapid transport of CSF in the subarachnoid space occurs by vascular pulsations causing mixing of CSF (Fig. 1b). Mixing is a diffuse, random process and the transport of CSF tracers occurs in all directions, down the concentration gradient of

the tracer. Although there are intraventricular pulsations causing mixing of CSF, the unidirectional bulk flow from the ventricles to the subarachnoid space dominates the flow of CSF in the ventricles. The main purpose of the CSF is to protect the brain from mechanical damage and serve as a cushion for the brain. The immersion of the brain into a liquid reduces its weight substantially (97%) and CSF dampens the effects of intra-cranial and extra-cranial forces. CSF also serves as transport vehicle for different neurotransmitters and other metabolites produced in the brain.

The choroid plexus produces 500 ml of CSF per 24 h and the total CSF volume is 120–150 ml. CSF is thus recycled over 3 times per day. The brain capillaries also produce a significant amount of fluid. The filtration and absorption of fluid in the brain capillaries is governed by the Starling principle. The interstitial fluid originating from the brain capillary adequately substitutes the CSF in the subarachnoid space, at intraventricular CSF obstructions. Homeostasis in the fluid surrounding the brain is thus maintained by exchange of the interstitial fluid across the thin arachnoid membrane covering the outer surface of the brain. Homeostasis is also maintained by capillaries in other organs [18] surrounded by fluid, i.e. fluid in the eye, pleura, peritoneum, pericardium and articulate joints. The total volume of the interstitial fluid is twice that of the CSF and the chemical composition of the fluids is similar. There is a rapid diffusion and mixing of CSF and interstitial fluid, aided by arterial pulsations, across the outer surface of the brain. This makes it impossible to separate the fluids despite their different origins. Thus, fluid outside the brain defines CSF and fluid within the interstitial space of the brain defines interstitial fluid. Although there are minor variations of the chemical CSF composition in different regions of the subarachnoid space, the CSF and interstitial fluid cannot be separated by differences in composition. The most characteristic feature of unbound brain water (CSF and interstitial fluid) is its low-protein concentration amounting to 0.4% of the protein concentration in plasma.

The intimate relation between the formation of CSF and interstitial fluid is also important for the absorption of CSF. Since there is net production of CSF in the choroid plexus, there is also a minute net absorption of CSF from the subarachnoid space into the brain and spinal cord (Fig. 3). The absorption of CSF by the brain capillaries occurs in the same way as capillaries in the other parts of the body absorb interstitial fluid. The brain capillary actively absorbs the macromolecules and plasma proteins in the CSF. One of the pivotal properties of the arteriole and brain capillary is to regulate and maintain fluid homeostasis at a normal slightly positive intracranial pressure (Fig. 3b).

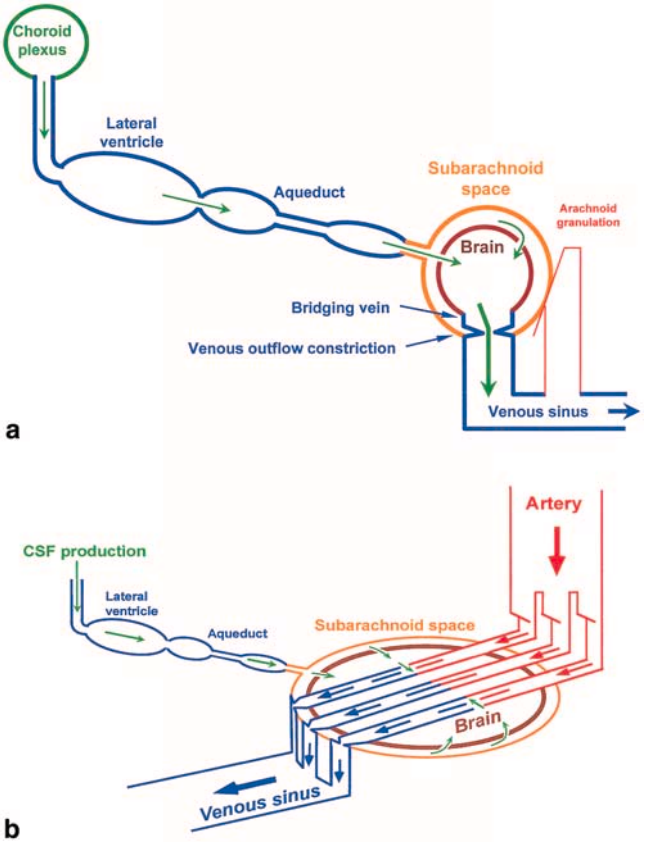

Fig. 3a, b Revised CSF circulation and its relation to cerebral blood circulation. **a** The brain capillaries absorb the CSF. **b** The absorption of CSF means a minimal addition to the major filtration and absorption of water through the brain capillaries. The intracranial pressure is regulated by filtration and absorption of fluid in the capillaries. The arterioles and capillaries regulate the intracranial pressure at a slightly positive level. This positive CSF pressure causes slight obstruction of the venous outlets, which explains the prompt pressure drop ("waterfall phenomenon") from the bridging veins to the venous sinus (**a**, **b**). The venous outflow resistance facilitates blood flow by distending the upstream-located cerebral veins and capillaries

Definition of hydrocephalus and transmantle pulsatile stress

The definition of hydrocephalus is enlarged ventricles at the expense of a narrowed subarachnoid space. This indicates the presence of an increased regional force directed from the ventricles towards the subarachnoid space, i.e. an increased transmantle pressure gradient. Hydrocephalus differs from processes lacking a transmantle gradient such as cerebral atrophy, where both the ventricles and subarachnoid space enlarge. The transmantle pressure gradient is very small. Studies, using direct intraventricular and subarachnoid pressure monitoring, have reported gradients ranging from 0 to 5 cm of water in hydrocephalus [19, 20]. Some studies could thus not disclose any transmantle gradient [19]. However, the transmantle pressure gradient exists as a dynamic phenomenon acting over time. Due to the law of Pascal, it can only exist transiently in a fluid-filled cavity and requires repeated pulse waves to persist [9, 10]. Since direct

pressure measurements have been equivocal, the transmantle pressure gradient should be renamed "transmantle pulsatile stress" as suggested by Guinane [21], to stress its dynamic nature and its dependence on the pulse wave [9, 10].

Most biological tissues are characterized by plasticity and therefore respond to local forces by local displacement, deformation and remoulding. Small pressure gradients may deform the brain, since it has a high plasticity. The deformation of the brain and the CSF spaces defines hydrocephalus. The brain is displaced towards the skull and the cortical gyri are often compressed or flattened. The transmantle pressure gradient is the only possible force, which could be responsible for such deformation. The normalization of the CSF spaces following shunting indicates that the transmantle pressure gradient can be narrowed or reversed, which further support that it really exists.

According to modern theories, the cumulative effect of many pulse waves slowly remoulding the brain is the cause of the ventricular enlargement in chronic hydrocephalus. The deformation of the brain and CSF spaces indicates the presence of local pressure differences. Computed tomography (CT) and MRI are excellent tools to demonstrate anatomy. By analysing the anatomy in hydrocephalus, it is thus possible to demonstrate the accumulated effect of small local pressure differences and of more general pressure differences like the transmantle gradient, which are undetectable at direct pressure measurement. Later, we will return to this simple concept of analysing pressure gradients by using CT and MR imaging, because it may increase the understanding of communicating hydrocephalus, and may give new insights on the hydrocephalic process in chronic obstructive hydrocephalus.

Normal intracranial hydrodynamics

Overview

The Monro–Kellie doctrine states that the total volume of the four main intracranial components, i.e. the brain, the CSF, the arterial and the venous blood, is constant and that any volume increase in one component causes a matching decrease in the other components [17]. The doctrine is a consequence of fluids being incompressible. The systolic expansion of the intracranial arteries is thus balanced by a matching expulsion of CSF through the foramen magnum and expulsion of blood from the veins into the dural venous sinuses (Fig. 4). The systolic expansion of the arteries compresses the venous outlets of the bridging vein and causes a systolic flow in the venous sinuses. The compression occurs at the venous outlets because the dural sinuses are, from a functional point of view, located outside the cranial cavity and the pressure drop is maximal at this site. The pulsating intracranial extracerebral arteries cause the CSF to flow back and forth in the spinal canal.

When the pulse wave enters the cranium there is an immediate increase in the CSF pressure that, according to Pascal's law, will be evenly distributed in the whole intracranial space. Consequently, intracranial pressure differences equilibrate rapidly. However, temporary pressure gradients arise during the cardiac cycle because flow effects cause shifts in the intracranial fluids. The flows and gradients are largest at the openings of the cranial cavity, i.e. at the foramen magnum and at the venous outlets into the venous sinuses.

The speed of the pulse pressure is inversely proportional to compliance. In a non-compliant cavity the transmission of pulse pressure occurs with the speed of sound (1540 m/s in water). Because the intracranial veins and the spinal thecal sac are compliant, the speed of the intracranial pulse pressure is decreased to 5 m/s, i.e. much slower than in a non-compliant cavity. The time for the intracranial pulse wave to pass from the arteries to the bridging veins is about 30 ms. Therefore, it is possible to monitor the effects and the timing of the pulse wave by flow-sensitive MRI, since its time resolution is about 15 ms. However, the speed of the blood flow is much slower. The mean transit time for the blood to pass from the cerebral arteries to the cerebral veins is 3.5 s, i.e. about four cardiac cycles.

This indicates that the CSF pulse pressure transmitted from the arteries to the outlets of the bridging veins has ample time to interact with cerebral blood flow. In fact, the venous backpressure from the compressed venous outlets dilates the upstream-located venous side of the capillaries at the same time as the arterial pulse wave dilates the arterial side of the capillaries. In this way, the brain capillaries are kept open and the total cerebral vascular resistance is decreased. Due to the backpressure from the compliant spinal thecal sac, the venous outlets are slightly compressed and the capillaries are kept open by venous backpressure during the entire cardiac cycle. The venous backpressure in combination with maintained diastolic flow by the windkessel mechanism causes a decreased vascular resistance and facilitates a high blood flow in the capillaries during the entire cardiac cycle.

Intracranial hydrodynamics is thus dependent on the compliance of the thecal sac and the compressible outlets of the bridging veins. Compliance is the capacity of a buffer system to accommodate a volume change and is defined as volume change divided by pressure change (dV/dP). It is a dynamic entity and cannot be measured directly since the system must be subjected to a volume change or a pressure change. Flow-sensitive MRI and intracranial pressure monitoring can measure intracranial volume and pressure changes induced by the arterial pulsations. Intracranial compliance can be determined by combining the two methods.

Dynamics in healthy individuals

Thus, flow-sensitive MRI studies give precise information of intracranial dynamics in all dimensions, both spatially and temporary. For the first time it is possible to

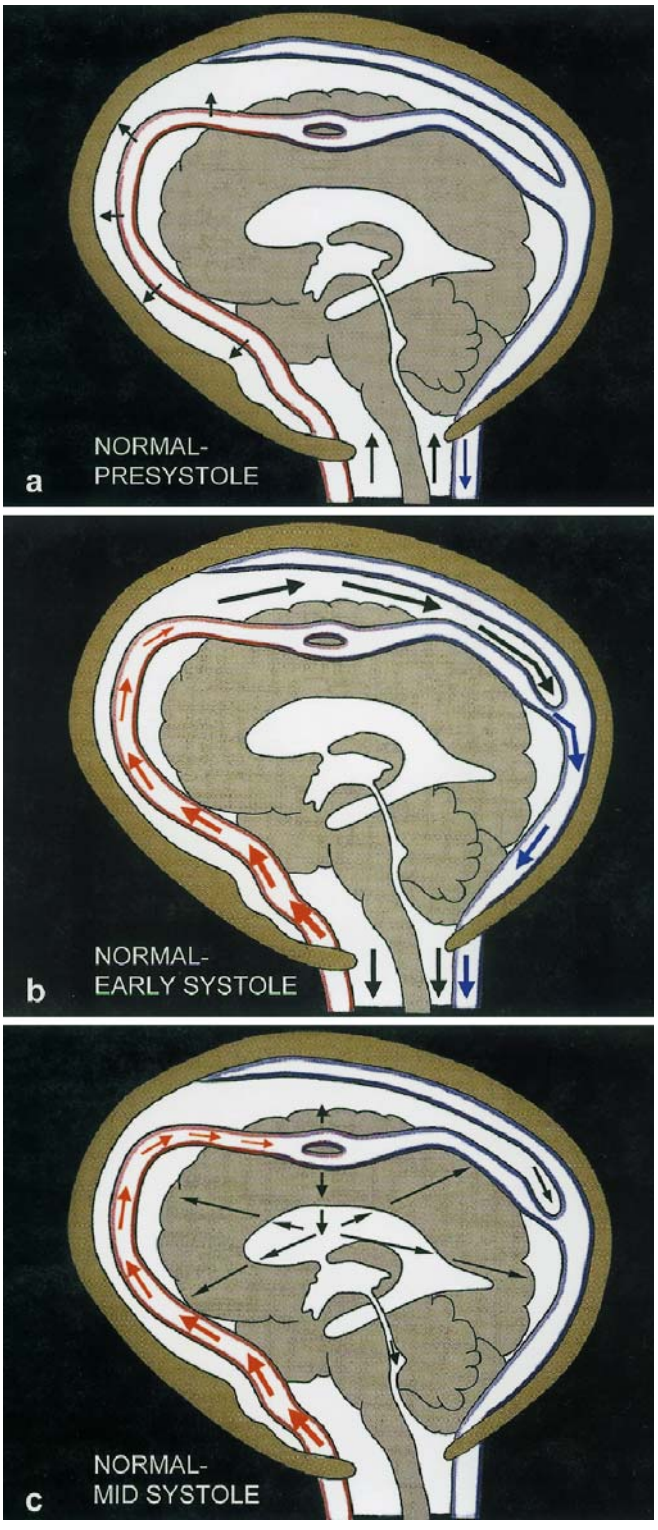

Fig. 4a–c Normal intracranial hydrodynamics. The relative thickness of the *arrows* in the artery indicates the magnitude of pressure. The relative thickness of the *arrows* in the venous system and subarachnoid space indicates the magnitude of flow. **a** *Presystole*. No pressure gradients are present in the brain. CSF is flowing upward into the cranial cavity from the compliant spinal canal. The normal venous outflow obstruction is not shown, for clearer demonstration of the changes occurring during systole. **b** *Early systole*. The systolic pulse wave causes a large expansion of the arteries with a concomitant and significant dampening of the arterial pulse pressure. The pressure is immediately transmitted to the entire subarachnoid space. The expansion of the arteries causes a large volume conduction of CSF that compresses the outlets of the cortical veins and increases the systolic blood flow in the venous sinus. Simultaneously the arterial expansion causes a large systolic expulsion of CSF into the compliant spinal canal. **c** *Mid-systole*. After some delay (60 ms), the small and dampened pulse wave in the artery (*thin arrows*) is transmitted into the brain capillaries. This causes a slight brain expansion and a transmantle stress of normal magnitude (modified from Int J Neuroradiol 1997;3:367–375)

differentiate the arterial expansion from the brain (capillary) expansion [6, 10, 22, 23]. The arterial expansion differs significantly from the brain (capillary) expansion both in timing and in magnitude. The volume increase of the intracranial extracerebral arteries is about 1.5 ml and equals the sum of the systolic stroke volumes at the foramen magnum (0.8 ml) and in the venous sinuses (0.7 ml) (Fig. 4b) [22, 23]. The systolic volume increase of the brain is about 0.03 ml. The minute brain expansion occurs *inwards towards the ventricular system* and equals the systolic stroke volume in the aqueduct (0.03 ml/beat) [22, 23]. The brain expansion is caused by the systolic expansion of the brain capillaries (Fig. 4c). The brain (capillary) expansion is thus only 2% of the arterial expansion. The arterial expansion starts 100 ms after the R-wave in the ECG complex and has a fixed duration of about 300 ms [7]. The brain expansion starts 60 ms later than the arterial one and occupies half of the cardiac cycle, i.e. 400–500 ms [7].

Of outmost importance to intracranial dynamics is the direct volume conduction of the pulse wave from the expanding extracerebral arteries, via the CSF, to the veins and spinal canal, thereby *bypassing* the brain and its capillaries (Fig. 4b). Arteries are compliant and act as an elastic reservoir. The expanding arterial wall absorbs part of the hydraulic energy in the pulse wave, which is released in diastole to maintain constant capillary flow. This is known as the "windkessel" mechanism. It transforms the pulsating arterial flow into a continuous and almost non-pulsating capillary flow. The windkessel effect of the arteries may be compared to a bagpipe where the elastic capacitance of the bag (artery) transforms the pulsatile inflow of air into a continuous non-pulsatile outflow thought the pipe (capillary). The presence of intracranial compliance, which allows the arteries to expand, is a mandatory prerequisite for the windkessel mechanism. The intracranial windkessel mechanism demonstrates large arterial expansion and almost absent capillary expansion [6], as described above.

Abnormal intracranial hydrodynamics

Overview

As realized by Dandy, an obstructed bulk flow of CSF at the pacchionian granulations cannot cause communicating hydrocephalus (Figs. 2d, 5a) Communicating hydroceph-

alus is instead caused by *decreased intracranial compliance*. Virtually all available hydrocephalus tests demonstrate decreased intracranial compliance or consequences of decreased compliance, especially when followed over longer periods. Most invasive tests interfere with intracranial compliance to some extent, since CSF infusion decreases compliance and CSF diversion increases compliance. The clinical improvement following CSF diversion, using the lumbar CSF tap test or shunt placement, is due to a forced compensatory dilation of the compressed intracranial veins. The forced dilation of the veins is a consequence of the Monro–Kellie doctrine, since successful shunting is based on a slight over-drainage of CSF, which must be compensated by a matching increase in venous and capillary blood volume. The dilated vessels increase intracranial venous compliance and cerebral blood flow. Shunting is thus not only a treatment, but may also be regarded as a test of restored venous compliance [10, 22] and restored cerebral blood flow [24]. Some clinicians, somewhat illogically, even define hydrocephalus depending on whether the patients improve by shunting or not. Intrathecal bolus and infusion tests demonstrate decreased intracranial compliance as well as increased outflow resistance of CSF through the compressed capillaries. As explained earlier, the outflow of the injected mock CSF occurs through the compressed cerebral capillaries and veins, not through the pacchionian granulations.

The expression of decreased compliance in communicating hydrocephalus is increased intracranial pulse pressure and/or decreased intracranial stroke volume. Thus, intracranial pressure monitoring demonstrates increased CSF pulse pressure as well as increased incidence of intermittent high-pressure waves of vascular origin, the so-called A and B waves. The elevation of the pulsatility index (PI) at transcranial ultrasound Doppler indicates increased pulsatility in major intracranial arteries [25, 26]. The increased pulsatility is a consequence of decreased intracranial compliance and breakdown of the windkessel mechanism, decreasing the diastolic flow in the arteries. Decreased intracranial compliance also increases the vascular impedance, i.e. increased resistance to pulsating flow, causing a decreased mean blood flow [6, 27, 28]. Decreased intracranial compliance has hitherto been a rather unknown cause for reduced cerebral blood flow. The compressed capillaries and veins also increase the vascular resistance to the convective blood flow [6]. Studies using flow-sensitive MRI report significantly decreased systolic stroke volumes of CSF and blood into the cervical spinal canal [22, 23, 29] and venous sinuses [23], i.e. decreased intracranial stroke volume.

The consequences of decreased compliance in hydrocephalus has until now not been fully appreciated. One reason for this may be that communicating hydrocephalus represents an unfamiliar condition where decreased compliance occurs at normal or near-normal intracranial pressure. Since the decreased intracranial compliance interferes with cerebral blood flow, it is conceivable that it may be responsible for the salient features of communicating hydrocephalus. In fact, the clinical signs and symptoms [5] as well as ventricular dilation, periventricular oedema, reduced cerebral blood flow, malabsorption of CSF, intracranial pressure waves, increase of mean CSF pressure, increased CSF pulse pressure, increased vascular resistance, hyperdynamic intraventricular CSF flow, increased PI and decreased intracranial stroke volumes may all be explained by decreased intracranial compliance [6, 28].

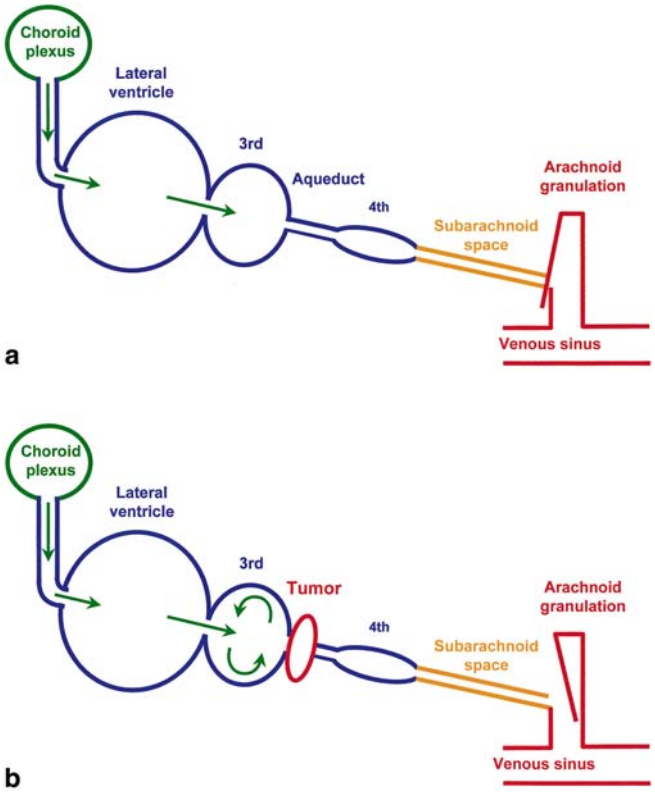

Fig. 5 a Communicating hydrocephalus is characterized by enlargement of the ventricles, mainly the lateral and third ventricle, and a narrow subarachnoid space. This anatomy cannot be explained by an obstruction to the bulk flow at the arachnoid granulations. It is obvious that we have to search for other explanations (cf. Fig. 6) than obstructed bulk flow of CSF as the cause of ventricular enlargement in communicating hydrocephalus. **b** Obstructive hydrocephalus is easy to understand, since the ventricles dilate proximal to an intraventricular obstruction to the bulk flow of CSF. An obstruction of the intraventricular foramen of Monro causes enlargement of the corresponding lateral ventricle, and so on. This schematic drawing shows a tumor in the aqueduct that dilates the third and lateral ventricles

Dynamics in communicating hydrocephalus

Flow-sensitive MRI in communicating hydrocephalus has shown that the stroke volume is decreased by 50% at the craniocervical junction [22, 23, 29] and by one-third in the venous sinuses [23]. This indicates that the total intracranial stroke volume in communicating hydrocephalus is about half that in normal individuals. Intracranial pressure monitoring reports a 6-fold increase of the CSF pulse

pressure as compared to normal individuals [30]. Intracranial compliance is thus decreased by 1 order of magnitude in communicating hydrocephalus, since compliance is the ratio between volume change and pressure change. This explains why the impact of the pulse wave may have such importance for the pathophysiology of communicating hydrocephalus.

Decreased intracranial compliance restricts the expansion of the arteries, causing breakdown of the windkessel mechanism with increased pulsations in the brain capillaries (Fig. 6) [10]. Since the artery cannot expand, there is decreased attenuation of pulse wave in the artery. The direct volume conduction from the artery to the bridging vein, which bypasses the brain capillaries, is decreased (Fig. 6b). Due to conservation of momentum, there must be a forced pressure and volume transmission of the pulse wave, from the artery into the capillary and brain tissue (Fig. 6c). The hydraulic energy of the pulse wave is absorbed in the capillary and the brain, instead of the artery. This causes an increased cerebral pressure as compared to the subarachnoid space and a diminished pressure difference between the vascular system and brain tissue.

The systolic pressure transmission from capillary to brain tissue increases cerebral venous pressure, diminishes perfusion pressure and decreases cerebral blood flow. It also diminishes the transcapillary pressure difference between blood and tissue, which decreases the fluid exchange across the capillary wall. Decreased intracranial compliance also increases the pressure transmission in the opposite direction, from the CSF to the vascular system. The pressures in the CSF and blood are closely coupled and equilibrate more rapidly at decreased intracranial compliance. Thus, an increase in CSF pressure does not sufficiently increase the pressure difference between CSF and blood, which is needed to absorb CSF. Since brain capillaries absorb the CSF, this is probably an important factor behind the malabsorption of CSF and behind the increased outflow resistance at CSF infusion tests. Other important factors for CSF malabsorption are decreased cerebral blood flow and increased vascular resistance in the compressed brain capillaries.

The increased brain expansion, directed *inwards* towards the ventricles [6, 10, 17, 22, 23] increases intraventricular pulse pressure and causes *a hyperdynamic CSF flow in the aqueduct*. The increased intraventricular pulse pressure in turn dilates the ventricular system and compresses the brain (Fig. 6c). This dynamic rebound phenomenon is not obvious and may seem counter-intuitive as discussed in the section of the transmantle pulsatile stress. The transmantle pulsatile stress may be explained by *self-compression of the brain against the ventricular system during each systole*, since ventricular fluid is incompressible and brain plasticity is high. Because of Pascal's law, the counter-force from the ventricles equals the force from the expanding brain. Due to its high plasticity, the brain does not fully resume its pre-systolic volume during diastole. The accumulated effect of systolic brain compression acting over time explains the compensatory enlargement of the ventricles. A similar pulsatile mechanism could explain the enlargement of subarachnoid cysts, since they are usually located in close connection to the large basal arteries. The cyst enlargement is equivalent to the enlargement of the Sylvian fissures in NPH.

The transmantle pulsatile stress compresses the capacitance vessels, i.e. the cerebral capillaries, cerebral veins and cortical veins. This increases vascular resistance and decreases cerebral blood flow (Fig. 7). The increased vascular resistance is also responsible for the small but significant increase in mean CSF pressure. Although the CSF pressure usually is within the normal limits in NPH, the mean pressure is significantly increased as compared to healthy individuals. The compressed capacitance vessels further decrease intracranial compliance and a vicious circle is formed. The mismatch between intracranial compliance and the blood circulation disturbs the normal autoregulation of cerebral blood flow [10] and increases the incidence of intermittent high-pressure waves. The regulation of the arterioles is grossly maintained, but arteriolar dilation causes increased pressure instead of increased cerebral blood flow. This explains the increased occurrence of the A and B high-pressure waves of vascular origin.

It is often necessary to combine clinical data with intracranial pressure monitoring or radiological follow-up studies to decide whether the hydrocephalic process is in an active or non-active state. Progressive ventricular enlargement is a sign of decreased compliance [31, 32] and enhanced transmantle pulsatile stress. It is not a sign of increased CSF pressure, since the pressure usually is near-normal, but the pressure can also be significantly increased. In fact, the volume of the ventricles is *inversely* dependent on the CSF pressure, statistically [33]. Therefore, it is impossible to predict the CSF pressure based on the ventricular volume in the individual case. Due to proximity to the compliant spinal sac, infratentorial compliance is usually less decreased than supratentorial compliance. This explains why the supratentorial ventricles may enlarge out of proportion to the fourth ventricle, which often is of normal or even small size (Fig. 5a). Consequently, obstructions to the pulsatile CSF flow in the prepontine cistern or in the cervical spinal canal are common when all four ventricles are enlarged. The decreased compliance in the posterior fossa increases the "transcerebellar pulsatile stress" and dilates the fourth ventricle.

To sum up, communicating hydrocephalus is a disorder of intracranial pulsations, caused by decreased compliance, [6, 9, 10, 22, 23, 28]. Decreased compliance restricts the arterial expansion and causes a breakdown of the windkessel mechanism. Therefore, communicating hydrocephalus has also been termed "restricted arterial pulsation hydrocephalus" [9, 10, 23]. Of outmost importance is the abnormal pressure and volume transmission into the brain capillaries, which increases ventricular pulse pressure, increases the pulsatile CSF flow in the aqueduct and dilates the ventricles.

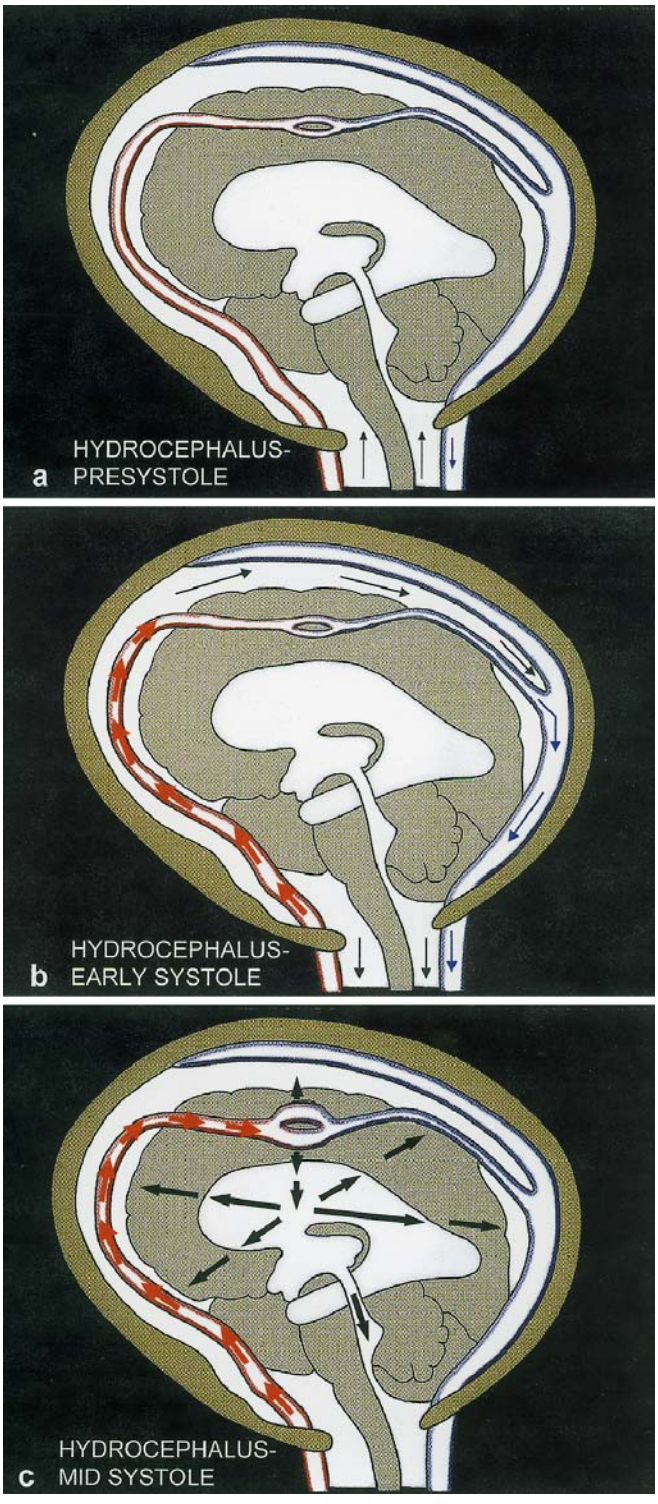

Fig. 6a–c Hydrodynamics of communicating hydrocephalus. Labels as in Fig. 4. **a** *Presystole*. The intracranial capacitance vessels are narrow and intracranial compliance is decreased. **b** *Early systole*. The decreased intracranial compliance restricts the arterial expansion, so little or no damping of the pulse pressure occurs in the artery. The artery behaves as if it were a rigid tube. Due to decreased volume conduction of CSF, the systolic flows in the dural sinus and in the subarachnoid space at the foramen magnum are decreased. The decreased arterial expansion causes a decreased volume conduction of CSF and decreased compression of the bridging veins at their outlets into the dural sinus. As a result, the venous outflow resistance is decreased and cannot dilate the upstream-located cerebral veins and capillaries any longer. The capacitance vessels are instead prone to collapse. The total vascular resistance is increased. **c** *Mid-systole*. The high pulse pressure in the artery (*thick arrows*) is transmitted undamped into the brain capillaries, giving rise to a large capillary expansion, an increased CSF pulse pressure, a large systolic flow in the aqueduct, an increased transmantle stress and ventricular dilatation. The increased transmantle stress compresses the intracranial veins in their entire length, which in turn decreases intracranial compliance, increases vascular resistance and decreases cerebral blood flow (modified from Int J Neuroradiol 1997;3:367–375)

Dynamics in obstructive hydrocephalus, acute phase

Any process that restricts the intraventricular bulk flow of CSF, e.g. an intraventricular block by mass lesion or adhesions, may cause obstructive hydrocephalus (Fig. 5b). The intraventricular block prevents the CSF to reach its main absorption site at the outer surface of the brain. The ventricles increase in volume because the periventricular capillaries can only absorb part of the CSF produced in the choroid plexus. The pressure in the ventricles and brain becomes elevated. The ventricular dilation displaces the surface of the brain towards the skull and compresses the cortical veins, mainly at their outlets close to the venous sinuses. This leads to venous congestion with increased blood volume and increased intracranial pressure.

Therefore, acute obstructive hydrocephalus has also been termed “venous congestion hydrocephalus” [9, 10]. The venous congestion and ensuing brain swelling counteracts the ventricular dilation, otherwise the ventricular dilatation would be fatal. At some point, a new equilibrium is achieved at higher pressure than normal. An important feature of acute hydrocephalus is the compression of the venous outlets, which is the main reason for the increased intracranial pressure. The venous outflow obstruction dilates the cerebral veins and capillaries upstream to the obstruction, which decreases the vascular resistance. This explains why the cerebral blood flow usually only is slightly decreased in the acute phase of obstructive hydrocephalus.

The arteriolar and capillary regulation of fluid absorption in the periventricular brain capillaries eventually balances the excess production of CSF from the trapped ventricle. The CSF pressure decreases and a new equilibrium is established at near-normal pressure in the chronic phase of obstructive hydrocephalus.

Dynamics in obstructive hydrocephalus, chronic phase

As described above, the CSF pressure decreases in the chronic phase of obstructive hydrocephalus. The CSF production and CSF absorption is balanced in the trapped ventricles. The venous outflow compression and venous congestion disappears. Consequently, there is no force to counteract the ventricular dilation any longer and the capacitance vessels become compressed. This decreases intracranial compliance. This condition exactly mimics the

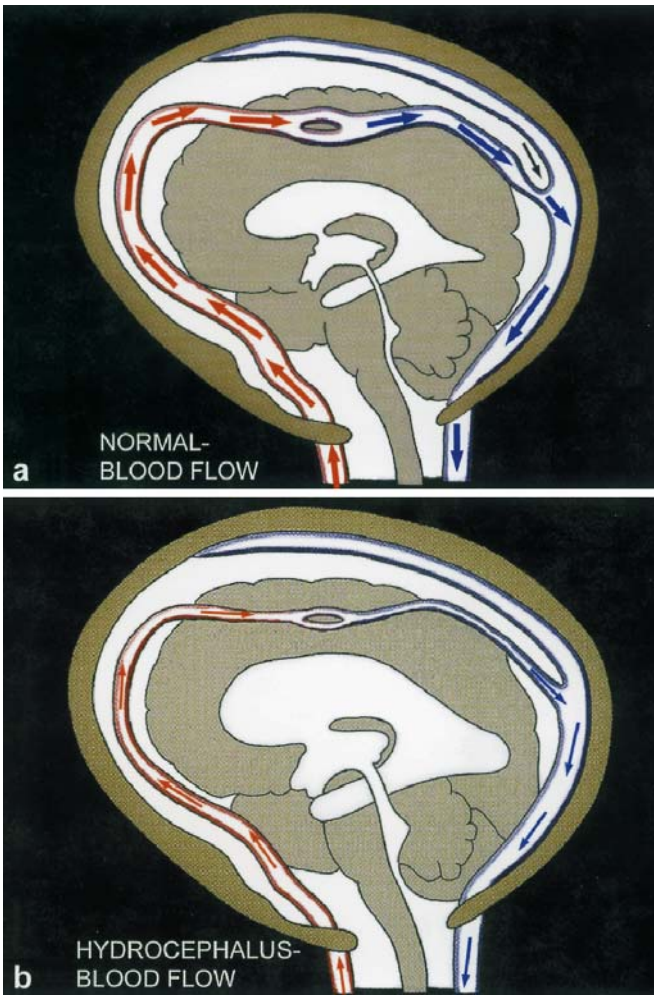

Fig. 7 Cerebral blood flow in **a** healthy individuals and in **b** communicating hydrocephalus. **a** The arterial windkessel mechanism, the wide intracranial vessels with small vascular resistance and the venous outflow resistance that keep the cerebral veins distended maintain the high normal blood flow. The venous outflow resistance is caused by a small positive intracranial pressure and is increased during systole. The venous outflow resistance is a mandatory prerequisite for the "waterfall phenomenon", i.e. the pressure drop occurring from the cortical veins to the venous sinus (cf. Fig. 3). **b** In communicating hydrocephalus, the increased transmantle pulsatile stress and the ventricular dilation compresses the cerebral veins and capillaries in their entire length. This significantly increases the vascular resistance and decreases the blood flow. The venous outflow resistance is reduced and the normal pressure drop from the subarachnoid space to the venous sinus (cf. Fig. 3) disappears. This phenomenon cannot be explained by the bulk flow concept, since a pressure drop caused by a CSF obstruction at the arachnoid granulations would increase the pressure drop from the subarachnoid space to the sinus, not decrease it. The reduced venous outflow resistance facilitates collapse of the compressed capacitance vessels, which further decreases cerebral blood flow

disorder of intracranial pulsations occurring in communicating hydrocephalus (Fig. 6). The decreased intracranial compliance causes a breakdown of the arterial windkessel mechanism with decreased arterial expansion, increased capillary expansion, increased CSF pulsations and hyperdynamic CSF flow in the trapped ventricle. The increased intraventricular pulse pressure increases the transmantle pulsatile stress, which continues to dilate the ventricles even if the mean CSF pressure is normal. MRI may disclose the hyperdynamic intraventricular pulsations by increased "flow void" in the foramina of Monro or in the aqueduct, within the trapped ventricles. Intracranial pressure monitoring demonstrates significantly increased CSF pulse pressure with normal or only slight increase of the mean CSF pressure [19]. Clinical signs and symptoms of NPH may develop, i.e. features identical with those in communicating hydrocephalus [5, 19, 34]. In support of this view, there is a clear-cut hyperdynamic CSF flow through the stoma following third ventriculostomy. The hyperdynamic flow in the stoma is equivalent to the hyperdynamic flow within the aqueduct in communicating hydrocephalus.

Intracranial dynamics following treatment (by third ventriculostomy, shunting or posterior fossa decompression) of chronic hydrocephalus and acute hydrocephalus

Chronic hydrocephalus

Since communicating hydrocephalus and chronic obstructive hydrocephalus share the same clinical symptoms, the same pathophysiology and can be treated by the same methods, it is logical to group these conditions under the heading of "chronic hydrocephalus". Shunting, third ventriculostomy and in some cases posterior fossa decompression are established methods to treat obstructive hydrocephalus. Shunting has hitherto been the only available method to treat communicating hydrocephalus, but recently there have been several preliminary reports of successful treatment by third ventriculostomy also in this condition [34–36]. This is not surprising when considering its similarity with the dynamics of chronic obstructive hydrocephalus as explained above. The primary aim of performing ventriculostomy or inserting a shunt in chronic hydrocephalus is not to increase the CSF absorption, but rather to increase intracranial compliance. The third ventriculostomy increases the systolic outflow from the ventricles, decreases the intraventricular pulse pressure and decreases the width of the ventricles. This would dilate the compressed capacitance vessels and increase intracranial compliance. The dilated capillaries facilitate increased blood flow and CSF absorption. Similarly, shunting restores venous compliance because the CSF diversion causes a forced dilatation of the compressed capacitance vessels.

Acute hydrocephalus

In acute obstructive hydrocephalus, a third ventriculostomy decreases the size of the ventricles and reduces the intracranial pressure by bypassing the intraventricular obstruction and re-establishing the bulk flow of CSF from

the ventricles to the subarachnoid space. Similarly, shunting reduces the intracranial pressure by diverting fluid from the trapped ventricles.

Posterior fossa decompression

Chiari malformations and other mass lesions at the craniocervical junction, which are thought to obstruct the CSF flow from the fourth ventricle, can be successfully treated by posterior fossa decompression. However, the hydrocephalus caused by Chiari I and Chiari II malformations is often of the communicating type. Achondroplasia also causes communicating hydrocephalus. All these conditions with communicating hydrocephalus can be treated by posterior fossa decompression. The underlying mechanism is that the posterior fossa decompression increases the intracranial compliance by recreating a communication with the compliant spinal thecal sac.

To sum up, acute hydrocephalus is characterized by venous outflow compression with dilated capacitance vessels. Chronic hydrocephalus is characterized by compressed capacitance vessels and decreased compliance. Thus from a clinical and physiological point of view, hydrocephalus could be divided into acute hydrocephalus and chronic hydrocephalus where the former is of obstructive type and the latter may be either of communicating type or of obstructive type. It is interesting to note that it is possible to treat acute as well as chronic hydrocephalus by shunting, third ventriculostomy or posterior fossa decompression. In the acute phase, it is important to reduce the intracranial pressure by draining fluid from the obstructed ventricles. In the chronic phase, it is important to increase intracranial compliance by dilating the capacitance vessels or by restoring communication with the compliant spinal canal.

Clinical symptoms of hydrocephalus

The clinical symptoms of hydrocephalus are a consequence of expansion of the ventricular system, raised intracranial pressure or decreased intracranial compliance. The clinical expression of these pathologies depends on the age of the patient. Raised intracranial pressure is the hallmark of acute obstructive hydrocephalus and may cause headache, nausea, vomiting, drowsiness and decreased consciousness. In communicating hydrocephalus, the symptoms are mainly caused by decreased intracranial compliance and decreased cerebral blood flow. Gait ataxia, mental deterioration, urinary incontinence and a normal CSF pressure characterize the clinical syndrome of NPH [5]. Not only communicating hydrocephalus but also benign aqueduct stenosis may cause the clinical syndrome of NPH [5, 34].

Radiological assessment of hydrocephalus

Overview

Cerebral atrophy is due to loss of brain tissue caused by degenerative disorders such as Alzheimer's disease or other types of brain damage [37]. Secondary to the loss of brain tissue, there is enlargement of the ventricles and the subarachnoid space. The cortical sulci are wide and the brain convolutions have a peg-like appearance. The perihippocampal fissure is dilated in Alzheimer disease, but not in NPH [38]. The temporal horns are more dilated in hydrocephalus than in cerebral atrophy [39, 40]. Another sign of hydrocephalus, easily demonstrated on axial and sagittal MR images, is the rounded shape of third ventricle with downwards bulging of its floor or dilation of the anterior and posterior recesses [41]. Although hydrocephalus demonstrates a narrow subarachnoid space with compressed and flattened brain convolutions, the differential diagnosis between communicating hydrocephalus and cerebral atrophy may sometimes be difficult. Therefore a more detailed description of the radiological findings and their underlying pathophysiology in communicating hydrocephalus are described below.

Anatomy of communicating hydrocephalus: other regional pressure gradients

Patients with communicating hydrocephalus, especially NPH, often demonstrate other changes than dilation of the supratentorial ventricles including the temporal horns. The brain is slightly compressed against the skull and the majority of the supratentorial cortical sulci are narrow. As explained earlier, the transmantle pulsatile stress is responsible for this deformation. The transmantle pulsatile stress is largest at the ventricular wall, which is interposed between the pulsating brain and the incompressible fluid in the ventricles. It is conceivable that this stress is most pronounced at the horns of the lateral ventricle explaining the occurrence periventricular oedema in these regions [42]. CT and MRI also demonstrate the effects of other regional pressure gradients than the transmantle gradient. The fourth ventricle is usually less dilated and may even be of normal or of small size. The Sylvian fissures and few regional sulci, which communicate with CSF spaces containing major cerebral arteries, are often enlarged [40]. An important sign often seen NPH is the pronounced compression of the upper part of the brain in the parasagittal region. The frontoparietal gyri are compressed and flattened and the sulci are narrow at the vertex, changes misinterpreted as due to the so-called "convexity block hydrocephalus" [40].

The subarachnoid space around the brainstem and cerebellum communicates widely with the compliant spinal canal. This maintains infratentorial compliance, which explains why the fourth ventricle usually is not dilated in communicating hydrocephalus. However, an obstruction at foramen magnum or adhesions in the basal

cisterns decreases infratentorial compliance, resulting in tetra-ventricular hydrocephalus.

The enlargement of the Sylvian fissures and regional cortical sulci indicates areas with increased CSF pulse pressure from the nearby-located arteries. The CSF pulse pressure is increased due to the decreased supratentorial compliance in communicating hydrocephalus. The repeated force from these systolic CSF pulsations compresses the brain. The dynamic pressure gradient in the Sylvian fissure is directed in the opposite direction of the transmantle gradient, i.e. from the subarachnoid space to the brain.

The compression of the upper part of the brain is most likely due to cranial displacement of the entire brain. Normally, the cortical subarachnoid space is widest in its parasagittal part of the convexity. In communicating hydrocephalus, this space is most compressed. This explains the finding of the "convexity block" at encephalography [40] and radionuclide cisternography [14]. The term "convexity block" describes adhesions or other obstructions to the CSF bulk flow at the convexity-vertex. However, the finding of a "convexity block" is usually not caused by pathological changes in the subarachnoid space [5]. It is also unrelated to the "bulk flow of CSF". Instead, the regional brain compression, causing a very narrow subarachnoid space, explains the lack of radionuclide tracer at the vertex. A cranial shift of the brain may also contribute to the impingement of the corpus callosum on the falx [43], where the posterior part of corpus callosum is cranially displaced against the falx. The cranial displacement of the two cerebral hemispheres also causes a v-shaped corpus callosum on coronal MR images [37].

Although increased pulsations of the arteries in the basal cisterns could explain the cranial displacement of the brain, it probably has another origin. In normal individuals, the cerebral veins are wide in relation to the blood flow and there is no flow resistance or pressure gradient in the vein. In communicating hydrocephalus, the cerebral veins and capillaries are compressed in their entire length. When the blood flows through the narrow vessels, it causes a significant pressure gradient in the venous system. To maintain flow in the narrow veins, the blood pressure must be significantly higher proximally in the intracerebral veins than distally in the bridging veins. This corresponds to a transvenous pressure gradient, directed from the brain capillaries to the venous outlets at the superior sagittal sinus. Since the majority of the superficial cortical veins drain into the superior sagittal sinus, the brain is displaced cranially along the transvenous gradient. The transvenous pressure gradient is a fixed and rather strong gradient maintained by the continuous blood flow in the narrow cortical veins. It explains the constancy of the "convexity block" at air encephalography and radionuclide cisternography and why it is impossible to fill the convexity-vertex region with air in communicating hydrocephalus [14, 40].

CSF diversion or shunting dilates the cerebral veins, which in turn increases intracranial compliance, decreases venous resistance and increases blood flow [24]. The increased blood flow improves the clinical syndrome. The restored compliance and reduced venous resistance decreases all intracranial pressure gradients and re-establishes almost normal anatomy. Widening of the upper parasagittal cerebral sulci and the subarachnoid space at the convexity is often a more sensitive sign of radiological improvement than the reduction of the third ventricle, lateral ventricles or temporal horns, following third ventriculostomy.

To sum up, transmantle pulsatile stress and other regional intracranial pressure gradients explain the typical CT findings in communicating hydrocephalus. The enlarged pulsating ventricles and CSF spaces behave like a slowly expanding mass. The brain is often compressed and displaced cranially by the transvenous pressure gradient, which explains the finding of the so-called "convexity block" at radionuclide cisternography as well as the compression of the brain at the convexity-vertex, demonstrated by CT and MRI.

CSF flow-sensitive MR imaging

It is very important to differentiate communicating hydrocephalus from obstructive hydrocephalus. MRI should be performed when CT or ultrasound are equivocal in this respect. When a tumour cannot be ruled out as the cause of an obstructive hydrocephalus, a contrast-enhanced CT or MR study should be performed. MRI evaluates the anatomy in the posterior fossa better than CT and ultrasound. Once the cause of hydrocephalus has been established, CT and ultrasound are excellent for following the size of the ventricles. As opposed to CT, MRI can detect and estimate the CSF flow in the narrow parts of the ventricles, i.e. the foramen Monro, the aqueduct and the outflows of the fourth ventricle. On routine T2-weighted MR scans, CSF flow can be detected by the flow-related signal loss or "flow void" in these areas of rapid and turbulent flow. In the majority of cases, it is sufficient to use sagittal T2-weighted standard spin echo sequences with 3 mm thickness and 0.5 mm gap, centred on the aqueduct. Since the 180° refocusing pulses in fast spin echo sequences are flow compensating, it is preferable to use the old-fashion dual echo sequence without flow compensation to detect CSF flow (Fig. 8). To decrease the scan time, a short repetition time (TR) of 1.5 s, 3/4 NEX and reduced spatial resolution can be used. This conventional dual echo sequence without flow compensation reliably demonstrates "flow void" in the region of the aqueduct or the third ventriculostomy secondary to pulsatile flow. Fischbein et al. found that modern sagittal 3-mm to 4-mm fast spin echo sequences were as sensitive as phase contrast MRI to examine the patency or occlusion of a third ventriculostomy [44]. Phase contrast studies are thus not needed in the routine evaluation of obstructions in the CSF pathways. Phase contrast MRI is a special technique that uses the relative phase angle of moving spins to quantify intracranial CSF flows. Sagittal phase contrast studies of CSF flow may be used as an adjunct

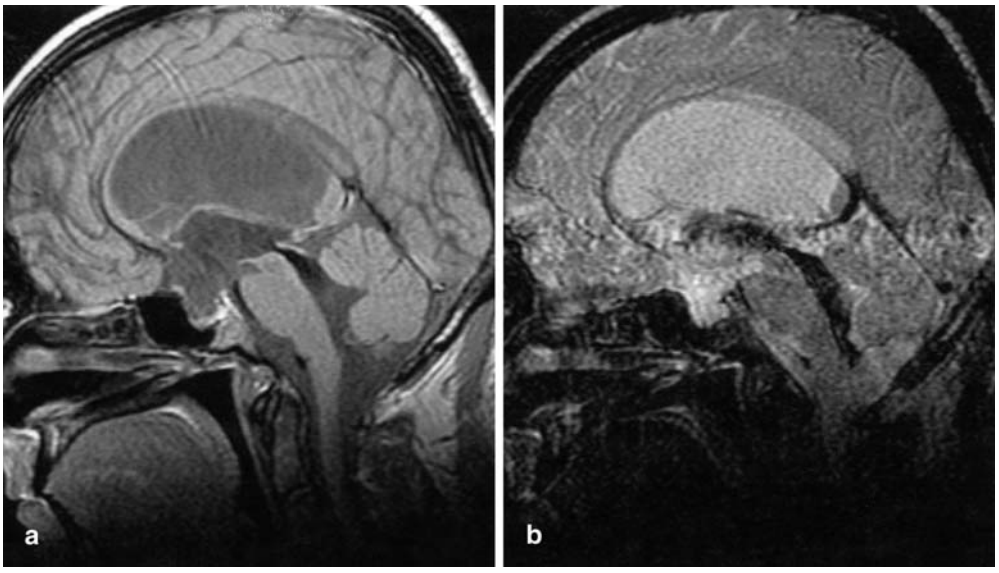

Fig. 8a, b *Aqueduct*: a standard dual echo sequence without flow compensation can be used to demonstrate or rule out intraventricular CSF obstructions. **a** The proton-density weighted image of this case of communicating hydrocephalus demonstrates the anatomy with widening of aqueduct. **b** The T2-weighted image shows a marked "flow void" in the aqueduct, the third and the fourth ventricle. This "flow void", i.e. loss of signal in the CSF, is caused by an increased pulsatile flow of CSF in the aqueduct during systole and diastole. The increased intraventricular CSF flow in turn is caused by an increased expansion of the brain capillaries during systole. The "flow void" sign is absent at obstructions to the CSF flow

diagnostic tool. Our group uses pulse-gated phase contrast studies in a plane perpendicular to the CSF flow, for complimentary radiological evaluation of CSF obstructions, and as a research tool for quantification of the stroke volume of CSF.

Prior to a third ventriculostomy, sagittal high-resolution and high-contrast T2-weighted sequences, like 3D constructive interference in steady-state (CISS) or 3D fast imaging employing steady-state acquisition (FIESTA), can be used to detect thin subarachnoid membranes in the ventricles and in the prepontine cistern [45, 46]. It is important to surgically penetrate adhesions and membranes such as Liliequist's membrane in the upper prepontine cistern to establish a free CSF communication to the compliant spinal CSF spaces. Liliequist's membrane is an arachnoid condensation extending from the upper border of dorsum sellae to the anterior edge of the mammillary bodies.

Causes of acute obstructive hydrocephalus

Obstructive hydrocephalus is caused by obstructions to the intraventricular flow of CSF, i.e. an intraventricular block by tumour or adhesions. Since the ensuing ventricular enlargement causes acute venous stasis, this condition has also been termed "venous congestion hydrocephalus" [9, 10]. Intraventricular bleeding may cause acute obstructive hydrocephalus that often resolves when the bleeding is absorbed [47, 48].

Causes of communicating hydrocephalus

As mentioned, communicating hydrocephalus as well as the other type of chronic hydrocephalus, i.e. chronic obstructive hydrocephalus, are caused by decreased intracranial compliance. Thus any process that restricts the arterial pulsations may cause chronic hydrocephalus, which consequently also has been termed "restricted arterial pulsation hydrocephalus" [9, 10].

The cause of communicating hydrocephalus is important, since it may provide a direct link towards its origin and thereby makes its pathophysiology easier to understand. Here I would like to emphasize the vulnerability of the subarachnoid space for various derangements that at first glance may seem insignificant. Meningitis, or even a small amount of blood following trauma, operation or aneurysm bleeding, may give rise to adhesions in the subarachnoid space that significantly reduces intracranial compliance. Intraventricular bleedings seldom causes chronic obstructive hydrocephalus, but may cause adhesions in the subarachnoid space resulting in chronic communicating hydrocephalus. An experimentally induced adhesion in the subarachnoid space is a stronger initiator of hydrocephalus than an intraventricular obstruction, especially if the basal cisterns are involved in the process [49]. Even a subtotal CSF blockage in the basal cisterns effectively restricts the fast expansion of the intracranial arteries. Dandy was unable to produce hydrocephalus by obstructing the aqueduct in cats since the intraventricular CSF production and CSF absorption is balanced in this species [1]. However, kaolin injection causing adhesions in the cisterna magna invariably result in chronic hydrocephalus, irrespective of the type of experimental animal used. Similarly, adhesions at the

convexity also cause hydrocephalus, because they obstruct the volume conduction of CSF from the expanding basal arteries to compliant veins at the vertex. Kaolin injection into the cranial part of the subarachnoid space produces hydrocephalus of a pure communicating type [50]. Thus, any process that interferes with the expansion of the arteries in the subarachnoid space may cause communicating hydrocephalus. That the bulk flow of CSF is an unimportant factor for the development of hydrocephalus is evident in cases of communicating hydrocephalus caused by obstructions to the pulsatile CSF flow at the craniocervical junction. Chiari I and II malformations, achondroplasia and intraspinal tumours may all cause communicating hydrocephalus, although the obstructions at or below the foramen magnum do not interfere with the intracranial bulk flow of CSF.

The so-called idiopathic communicating hydrocephalus is not characterized by any changes in the subarachnoid space [5]. Does this indicate that a pathogenesis is lacking in this common category? It is highly unlikely that the distinct features of communicating hydrocephalus, i.e. clinical symptoms, ventricular dilation, increased pulse pressure and transmantle pulsatile stress, could occur without an underlying pathogenesis. Instead, it is conceivable that any vascular disorder, decreasing arterial compliance and increasing capillary pulse pressure, is a common cause for the so-called idiopathic communicating hydrocephalus. It is a well-known fact that communicating hydrocephalus is strongly associated with vascular disease [5, 51, 52]. Arterial hypertension, arterial ectasia, cerebral arteriosclerosis, small vessel disease of Binswanger type, diabetic microangiopathy, white matter lesions, old age and vascular spasm due to subarachnoid hemorrhage are all associated with communicating hydrocephalus [5, 9, 10, 22, 23, 51, 52]. It seems appropriate to use the term "restricted arterial pulsation hydrocephalus" or "increased capillary pulsation hydrocephalus" to stress the vascular pathophysiology of communicating hydrocephalus.

Treatment options in hydrocephalus

The optimal treatment for a patient with hydrocephalus is to remove the cause of hydrocephalus to restore normal flow of CSF and normal hemodynamic conditions in the central nervous system. Treatment aimed at removing the cause of hydrocephalus will often concentrate on the removal of tumours or other masses like arachnoid cysts. Posterior fossa decompression is a causative treatment of communicating hydrocephalus originating from obstructions to the pulsatile flow at the foramen magnum.

There are two main options for treatment when the cause of the obstructive hydrocephalus is not accessible for treatment, shunting and third ventriculostomy [53]. If the obstruction to CSF flow is found within the ventricular system or at the outflows from the fourth ventricle, a third ventriculostomy may successfully treat an obstructive hydrocephalus. The procedure is usually carried out using a neuroendoscopic technique by which the thin floor of the third ventricle is opened, thus creating a communication between the third ventricle and the suprasellar cisterns. The patency of such a third ventriculostomy can be checked using MRI as the pulsatile flow at the stoma can be seen on flow-sensitive sequences [44, 45]. The primary aim of the treatment in acute obstructive hydrocephalus is to decrease the size of the ventricles and restore a normal intraventricular pressure. Inserting a CSF-shunt, performing a third ventriculostomy, or in some cases a posterior fossa decompression, diverts the excess volume of CSF produced within the trapped ventricles, restoring anatomy and CSF pressure. The reduction of the intracranial pressure also causes a secondary increase of intracranial compliance.

The primary aim of the treatment in chronic obstructive hydrocephalus or communicating hydrocephalus is to restore intracranial compliance. The diversion of CSF following shunting causes a forced dilation of the compressed cortical veins. This increases venous compliance, decreases vascular resistance and increases cerebral blood flow. An alternate treatment is a third ventriculostomy [34–36]. This surgically created opening into the subarachnoid space ultimately reduces the intraventricular pulse pressure, due to increased expulsion of ventricular CSF during systole. This in turn will reduce the transmantle pulsatile stress, reduce ventricular size and expand the subarachnoid spaces including the compressed cortical veins, thus again restoring intracranial compliance and cerebral blood flow. In posterior fossa decompression, intracranial compliance is restored simply by recreating communication with the compliant spinal canal. Consequently, shunting, third ventriculostomy and posterior fossa decompression all increase intracranial compliance and decrease the transmantle pulsatile stress, which is causing the ventricular dilatation in chronic hydrocephalus.

The dynamic view on hydrocephalus presented here offers a logical explanation for the successful treatment of communicating hydrocephalus by third ventriculostomy and posterior fossa decompression. It cannot be explained by the CSF bulk flow theory, because a third ventriculostomy or posterior fossa decompression cannot influence a "decreased absorption of CSF at the pacchionian granulations". This supports the view that communicating hydrocephalus is not caused by decreased absorption of CSF, but rather by decreased intracranial compliance. The indications for treating communicating hydrocephalus at Chiari I malformations by posterior fossa decompression is well established and is now the preferred method. The indications for treatment of communicating hydrocephalus by third ventriculostomy have not yet been fully tested and remain to be determined [35, 36]. It seems that the clinical success rate for treatment by third ventriculostomy is somewhat lower in young children below the age of 1 year [54–56] and perhaps also for patients more than 60 years old. Third ventriculostomy offers an alternate treatment to shunting and vice versa. It may also be used to replace the shunt when a child is older. This strategy has been successful in shunted premature children with post-haem-

orrhagic hydrocephalus [56]. In this way, many children may become shunt independent. In this context, it should be pointed out that once patients have been treated by shunting or third ventriculostomy, they should be closely followed clinically. The reason for this is that chronic hydrocephalus is a dynamic disease and there are reports of sudden deterioration, which exceptionally could be fatal, after minor head trauma as well as following a late closure of the shunt or stoma [57].

The diagnosis and treatment of hydrocephalus is based on a combination of the clinical picture and the radiological findings. It is important that an incidental finding of hydrocephalus is described, since the patient and referring physician may overlook the clinical symptoms. There are reports of clinically not sufficiently examined "asymptomatic hydrocephalus" or so-called "chronic arrested hydrocephalus" showing significant improvement after treatment [58]. Considering the complexity of the mechanisms behind communicating hydrocephalus and that shunting only is a symptomatic treatment, the prediction of responders will continue to be most challenging. Until now no single test exist that can predict the surgical outcome. The clinical evaluation will probably remain an important prognostic tool [4, 5].

Discussion: brief review of hydrodynamic investigations and theories

The hydrodynamic theory presented here offers a logical explanation for most salient features of hydrocephalus. Due to its adaptability, it is usually possible to reinterpret previous hydrodynamic investigations in accordance with the new theory. The most significant objection to the theory originates from investigations demonstrating *increased compliance in chronic hydrocephalus* [59, 60]. It is likely that some of these findings are due to methodological imperfections, because decreased compliance has also been found by using the same method [32, 61]. The compliance is estimated by the slow variation of the mean CSF pressure following intrathecal injections of fluid [32, 62]. It is clear that these experiments are unable to detect the rapid variations of CSF pressure related to the cardiac cycle [63, 64]. Furthermore, the bolus injection technique and other intrathecal infusion methods that measure regional compliance are often too dependent on the absolute CSF pressure. The results from a study of chronic obstructive hydrocephalus in dogs illustrate this inaccuracy of the method [65]. At a mean CSF pressure of 10 mmHg, the compliance in hydrocephalic dogs was significantly increased as compared with normal dogs. However, the compliance was significantly decreased in the same hydrocephalic group at a mean CSF pressure of 20 mmHg. A test producing such disparate results is questionable and raises the question whether compliance tests should be performed at an increased CSF pressure [66, 67]. This also explains why infusion tests exceptionally may demonstrate increased compliance in hydrocephalus. It seems indicated to revive the thorough analysis of the CSF pulse wave made by Foltz [63, 64], i.e. to estimate the amplitude and rise time of the CSF pulse pressure during intracranial pressure monitoring. The systolic amplitude of the CSF pulse pressure is significantly increased and its rise time is significantly decreased indicating decreased intracranial compliance in chronic hydrocephalus [63, 64]. In this context it should be stressed that chronic hydrocephalus is a disorder of intracranial vascular pulsations and that the arterial pulse wave is a much better input function than intrathecal fluid injections when testing the compliance of the vascular system [6]. This notion is supported by the results of Foltz [63, 64] and Gonzales-Darder [68], which showed a significant correlation between the CSF pulse amplitude and intracranial compliance.

Only a few of the numerous MR studies of aqueductal CSF flow will be mentioned. Experimental investigations supporting the new theories will be discussed first. Then a short overview and comments to the early and recent MRI-based hydrodynamic theories will be given.

Animal studies supporting the new theory

In 1978, Di Rocco et al. introduced a small pulsating balloon into the lateral ventricle in lambs [69]. The increased ventricular CSF pulsations from the pulsating balloon caused communicating hydrocephalus in all animals, although the mean ventricular CSF pressure was normal. The experiment was inspired by O'Connell's idea that increased ventricular CSF pulsations could cause communicating hydrocephalus [2] and by Bering's suggestion that increased pulsations in the choroid plexus could act as a "water hammer" pulse within the ventricles [70]. This is the first experimental proof that increased CSF pulse pressure can cause hydrocephalus and that ventricular enlargement may develop without CSF obstruction and without increase of mean CSF pressure.

In 1977, Guinane produced regional communicating hydrocephalus in the olfactory ventricle of rabbits by injecting silicone rubber into the surrounding subarachnoid spaces [21]. He concluded that increased pulsatile stress at the ventricular wall is responsible for the ventricular dilation. The obstruction in the subarachnoid space decreases the arterial and venous compliance in the subarachnoid space. The restricted arterial pulsations in the subarachnoid space should increase the capillary expansion and transmantle pulsatile stress, which also is a hallmark for communicating hydrocephalus in humans. The regional ventricular dilation in rabbits occurred without CSF malabsorption and without increase of mean CSF pressure. This is conceivable since CSF malabsorption and increased mean pressure are secondary phenomena to the more general vascular disturbances occurring in human hydrocephalus. Furthermore, there is no choroid plexus in the olfactory ventricle. This supports the notion that it is the increased pulsations from the brain capillaries, rather than those from the choroid plexus, that are responsible the ventricular enlargement. The combined

findings of Di Rocco and Guinane thus support the new hydrodynamic theory on communicating hydrocephalus presented here.

Early hydrodynamic theories

In 1962, Bering challenged the traditional concepts on hydrocephalus by producing asymmetric hydrocephalus in dogs after cisternal kaolin injection and removal of the choroid plexus from one lateral ventricle [70]. The ventricle without choroid plexus remained small although there was communication between the two lateral ventricles. He concluded that the ventricular enlargement depended on undamped choroid plexus pulsations rather than increase in mean CSF pressure, proximal to the obstruction, as Dandy [1] had assumed. Although Bering confirmed O'Connell's view [2] that increased intraventricular pulsations could cause hydrocephalus, Bering's challenging concept has not gained general acceptance. One reason for this is that removal of the choroid plexus has not proved to be an efficient treatment of hydrocephalus. Using ultrasound, White et al. [71] demonstrated that the brain expansion occurred caudally and medially towards the lateral ventricles in normal individuals. Feinberg and Mark [72] using cardiac gated MRI confirmed a systolic compression from above towards the lateral ventricles. Later Poncelet, Enzman and Greitz using MRI phase studies verified that the brain expands inwards towards the ventricular system [17, 73, 74]. This, of course, excludes the choroid plexus as the main cause of the intraventricular CSF pulsations. Such systolic CSF pulsations would enlarge the ventricles rather than compress the ventricles.

In 1979, White et al. launched a completely new hydrodynamic concept [71]. Based on their ultrasound studies showing brain expansion towards the ventricles, they proposed that communicating hydrocephalus develops because of limitation of the pulsatile flow in the aqueduct. The intracranial hydrodynamics is thoroughly discussed in this extensive paper. Their theory, as well as the hydrodynamic theory presented here, preceded the successful treatment of communicating hydrocephalus by third ventriculostomy [35]. Foltz, being inspired by Bering, stressed the significance of the intraventricular CSF pulsatility as the cause of hydrocephalus in 1981 [63, 64]. He showed that the power of intraventricular CSF pulsations was increased up to 4 times in chronic hydrocephalus. He was also first to state, "In any progressing hydrocephalus, the high pulse pressure must reflect a loss of intracranial compliance based primarily on a loss of intracranial venous volume venting capability". In 1984, Sklar and Linder [32] suggested that increased brain elasticity could cause hydrocephalus and seriously questioned CSF malabsorption as a causative factor. CSF infusion tests always demonstrate a so-called "absorptive reserve" even in patients with most severe absorptive defects [32]. They also found that the elasticity sloop parameter and the CSF pulse amplitude correlated to the size of the ventricles [31, 32].

Recent MRI based hydrodynamic theories

In 1985, Bergstrand et al. performed the first dynamic MRI study of aqueductal CSF flow [75]. In 1986, Bradley demonstrated hyperdynamic CSF flow by "flow void" in the aqueduct in patients with communicating hydrocephalus [76] and later he linked this finding to whether the disorder is shunt-responsible or not [77, 78]. The hyperdynamic flow in the aqueduct has been verified in a number of MRI studies, but not in all [79, 80]. The finding of a hyperdynamic CSF flow supports the diagnosis of communicating hydrocephalus, but its absence does not rule out a shunt-responsible hydrocephalus. This is understandable since an increased ventricular pulse pressure may not cause a hyperdynamic flow in a narrow aqueduct, due to the increased CSF flow resistance. In treatable NPH, due to aqueductal stenosis, there is by definition no flow through the aqueduct. However, after third ventriculostomy a clear-cut hyperdynamic CSF flow in the stoma is the rule. This shows that CSF pressure monitoring, in addition to estimation of CSF stroke volumes, may be needed to make a complete evaluation of intracranial dynamics. It is also important to realize that the increased stroke volumes in the aqueduct is a paradox due to the redistribution of the intracranial pulsations occurring in chronic hydrocephalus. Decreased stroke volumes, not increased stroke volumes, characterize decreased compliance at a constant pulse pressure. In a cranial cavity with *no* compliance, the decreased intracranial stroke volumes in the venous sinuses and at the craniocervical junction would decrease further and even the pulsatile flow in the aqueduct would cease. Therefore, a small pulsatile flow in the aqueduct does by no means rule out decreased intracranial compliance.

In 1993, the hydrodynamic theory presented here was introduced [6] and was further developed in subsequent papers [9, 10], including the present paper. It should be emphasized that the theory is compatible with most of the observations made at previous experimental and clinical investigations and, like all theories, that it continuously needs modifications to fit reality. However, hardly any important corrections of the original basic theory have been made until now. The critique has been modest and no serious objections to the theory have been raised. The lack of critique has had an untoward side effect; the theory has not yet gained widespread acceptance. This may be due to its complexity, making the full consequences of the theory difficult to understand, but it may also be a consequence of the very slow acceptance rate of new ideas in medicine. It is 90 years since Dandy found that capillaries absorb the CSF and 60 years since O'Connel suggested increased CSF pulsations to be the cause of communicating hydrocephalus. Here I would like to elucidate an unclear point, which has been the subject for some misunderstandings. In 1997, I suggested venoconstriction as

medical therapy for communicating hydrocephalus [9]. The theoretical background was to refill the compressed brain capillaries, by venous backpressure, to regain capillary compliance. I did not stress that the therapy preferably should constrict the venous *outflows* and cause *dilation* not only of cerebral capillaries, but also of the upstream-located cerebral veins. Another positive effect of a balanced venous outflow constriction would be decreased total vascular resistance and increased cerebral blood flow [6, 9, 10], as commented upon in the end of this discussion. However, manipulations of the venous outflows should only be performed experimentally since an unbalanced constriction could cause venous congestion and increased intracranial pressure that even could be fatal.

In 2000, Bateman studied the flow in the basal intracranial arteries and in the superior and straight sagittal sinuses in patients with NPH by using MRI [81–83]. His findings support Foltz's notion that decreased compliance of the superficial cortical veins is a causative factor in communicating hydrocephalus [63, 64]. Although Bateman's main conclusion is correct, there are some misconceptions resulting in questionable speculations, which could have been avoided if he had studied previous writings on this subject more thoroughly. Bateman was first to directly measure the pulsatility in the cortical veins by MRI [83]. He found a decreased pulsatility in the cortical veins in hydrocephalus, which he interpreted as the result of compressed cortical veins decreasing cortical venous compliance. Interestingly, the present author predicted decreased stroke volumes within the cortical veins in hydrocephalus when analysing the pulsatile flow in the superior sagittal sinus [6, 22, 23]. The mechanism behind this is the decreased blood volume in *all capacitance vessels* in communicating hydrocephalus. The decreased venous blood volume does not only decrease the *extracerebral* volume conduction from the expanding intracranial extracerebral arteries to the venous outlets as explained earlier. The decreased blood volume in the intracerebral veins also decreases the volume conduction that occurs normally *within* the brain [6, 22]. This means that the normal *intracerebral* volume conduction from the expanding intracerebral arteries via the brain tissue to the intracerebral veins, thereby again bypassing the brain capillaries, is decreased in communicating hydrocephalus. The decreased *intracerebral* as well as the decreased *extracerebral* volume conduction causes a breakdown of the windkessel mechanism with increased expansion of the capillaries, in chronic hydrocephalus. This also argues against a common misconception, i.e. that structural changes within the brain tissue, itself, decreases cerebral compliance in chronic hydrocephalus. Instead, it is more likely that the decreased cerebral blood volume is the cause of the decreased cerebral compliance. This in turn may explain why the brain damage often is less significant after treatment of acute obstructive hydrocephalus than after treatment of chronic hydrocephalus. The venous congestion following acute hydrocephalus increases the cerebral blood volume, which in turn maintains cerebral compliance and protects the brain from adverse effects from the increased pulse pressure. The increased cerebral blood volume dampens the arterial pulse pressure by venous venting and acts as an "air bag" for brain tissue.

In 2001, Egnor et al. introduced a very attractive model of normal CSF pulsations based on a simple electrical circuit with capacitance, inductance and resistance elements [27]. Their model is to a great extent based on the hydrodynamic concept presented here and was also tested on different intracranial disorders including communicating hydrocephalus [28]. They stressed the importance of a normal compliance and normal windkessel mechanism as well as the importance of resonance within the cavity to maintain normal intracranial dynamics. According to the authors, disturbances in one or more of these parameters would lead to hydrocephalus. Although Egnor et al. adopted Bering's theory of increased choroid plexus pulsations as the cause of ventricular dilation, their model is also directly applicable on the hydrodynamic theory of hydrocephalus presented here. Arguments against Bering's theory have already been given in this discussion. It is fair to conclude that the papers of Egnor and the present paper mutually support each other.

Comment to the hydrodynamic theories and to the cause of ventricular dilation

Common to all hydrodynamic theories on chronic hydrocephalus is the fundamental importance of the increased intracranial pulse pressure and thus a decreased intracranial compliance. Can the mechanism of ventricular enlargement be explained in the light of this observation? Why do the ventricles and not the subarachnoid space enlarge in hydrocephalus? The solution to this enigma may be found in the results from MRI studies enabling a separation and quantification of the arterial and capillary expansion. In communicating hydrocephalus the expansion of the capillaries is significantly increased. The expansion thus originates within the brain and is also directed centripetally towards the ventricles. How can this asymmetrical brain expansion be explained? Due to the law of Pascal, the pressure is equal and no pressure gradients exist in the cranial cavity, before the arrival of pulse wave. This means that the pressure in the ventricles and subarachnoid space is equal. A movement preferable occurs towards the direction with least resistance. The hydrodynamic force to move an object against a resistance is inversely related to area of the object. The area of the ventricles is more than an order of magnitude less than the outer surface of the brain. Thus, for hydrodynamic reasons the systolic movements and the resulting stress on the brain must be much larger at the ventricular interface. As explained earlier, the counter-force from the non-compliant fluid in the ventricles on the plastic brain tissue causes a self-compression of the brain and dilation of the ventricles. The ventricular enlargement can thus be explained by an increased brain expansion and by the simple fact that the ventricular brain surface is smaller than the arachnoid brain surface. This also explains the

limitation of the hydrocephalic process. When the ventricles dilate, the difference between the two surfaces diminishes. Since the brain movements are inversely dependent on the area, the brain movements on the ventricular side will decrease and the asymmetric process that drives the dilation will stop.

Another way to explain the asymmetric brain expansion is to look upon the ventricular system as an invagination of the subarachnoid space. When brain volume increases, either its convex outer-surface must increase or its concave inner-surface towards the ventricles must decrease. The elastic properties of the brain restrain dilation of the outer surface, thereby counteracting outward expansion and facilitating inward expansion. This mechanism can be demonstrated by allowing a balloon to reexpand after having focally compressed (invaginated) it on one point or by the rapid expansion of the folding when a parachute is released. This type of hydromechanics is also used to empty the urinary bladder by increasing the abdominal pressure, the bladder being an invagination within the abdominal cavity. The sylvian fissure is a true invagination of the subarachnoid space. Increased systolic brain expansion in this invagination, in combination with increased pulse pressure from the middle cerebral artery within this space, may contribute the dilation of the sylvian fissures as seen in NPH [40]. This supports the view of O'Connel [2] and Bering [70] that a pulsating CSF space acts as a slowly expanding mass. It also explains why subarachnoid cysts located near pulsating intracranial arteries or pulsating CSF spaces may expand. An interesting movie made by Ertl-Wagner et al. clearly demonstrates that the brain expansion preferable occurs towards the lateral ventricles and sylvian fissures in healthy volunteers [84]. After suddenly increasing the intracranial venous pressure during the Valsalva maneuver, they were able to produce brain expansion by forcing the intracerebral veins and capillaries to expand in healthy volunteers, but not so in patients with NPH. Using MRI, they measured the ventricular volume before, during and after Valsalva maneuver. During Valsalva, the volume of ventricles decreased by as much as 18% in healthy volunteers. In an accompanying movie available on line at the Springer Link server, there is a clear volume decrease of ventricles and the sylvian fissures during Valsalva. An even more interesting finding in this study was that the volume decrease of the ventricles in patients with NPH was 0% during Valsalva. The reader is recommended to read this paper and look at these movies, since this is the first examination that visually demonstrates the striking effect of decreased intracranial compliance in communicating hydrocephalus. Even the most dedicated supporter of the bulk flow theory ought to consider decreased compliance as a causative factor in communicating hydrocephalus.

As evident from this review, the implications of the new hydrodynamic theory are numerous. If correct, extensions of the theory may influence, not only the understanding of the hydrocephalic process and normal cerebral vascular physiology, but also shed new light on the normal and abnormal vascular functions in the rest of the body. There are two general vascular observations that are of particular importance. The first is the active absorption of plasma proteins and macromolecules by the capillaries. Based on this mechanism a new hypothesis has been proposed implying the main absorption of plasma proteins in tissue occurs by the capillaries of the body, and not by lymphatic system as generally thought [18]. The other general vascular observation that needs to be explored further is the phenomenon of *dynamic negative flow resistance* occurring in veins and capillaries [6, 10, 23]. A prerequisite for the "dynamic negative resistance" is the venous outflow compression or "the waterfall phenomenon" occurring in all encapsulated organs (Fig. 3). Increased organ pressure increases the compression at the venous outflows, which paradoxically *decreases the total vascular resistance*. This corresponds to the hydromechanic phenomenon of "dynamic negative resistance" occurring when flow is present in a collapsible tubing and the pressure inside the tubing is equal or slightly higher than outside the tubing [85]. Veins and capillaries are collapsible and their intraluminal pressure is only slightly higher than the pressure in tissue. The reason for the decreased total flow resistance is that the venous backpressure, caused by the outflow obstruction, dilates the upstream-located and partly collapsed veins and capillaries. Although the venous outflow resistance is increased, the increased flow conductance in the upstream-located veins and capillaries exceeds the outflow resistance and causes a reduction of the total flow resistance. This indicates that, within the normal physiological pressure variations, there is *passive venous autoregulation* of the blood flow in most encapsulated organs [9, 10]. The regulation is passive, since the capacitance vessels dilate passively to the raised tissue pressure, which in turn increases the outflow resistance. The blood flow can be maintained at a higher tissue pressure although the perfusion pressure actually decreases, i.e. there is a dynamic negative flow resistance in the veins and capillaries. In chronic hydrocephalus, the compression of the capacitance vessels causes a breakdown of the "passive venous autoregulation" that in turn causes a breakdown of the normal arteriolar autoregulation of cerebral blood flow. Due to the increased flow resistance in the compressed capacitance vessels, the blood flow cannot increase despite maximal arteriolar dilation. This results in decreased cerebral blood flow and increased occurrence of intracranial high-pressure waves. It is likely that the phenomena of "dynamic negative venous resistance" and "passive venous autoregulation" could be tested in an electrical circuit model similar to that of Egnor and co-workers [27, 28].

Conclusions

That an intraventricular CSF blockage causes acute obstructive hydrocephalus is generally accepted. However, the mechanism behind communicating hydrocephalus,

especially NPH [4, 5], has been an enigma ever since their first description. The new theories presented here demonstrate that the underlying mechanisms follow basic hydromechanics familiar to any engineering student. All the significant features of communicating hydrocephalus can be explained by increased transmantle pulsatile stress affecting the brain, CSF spaces and vascular system. Communicating hydrocephalus is caused by decreased intracranial compliance and not by obstruction to the bulk flow of CSF. Decreased compliance causes an increase of the intracerebral pulse pressure, which increases the transmantle pulsatile stress and dilates the ventricles. The intracranial compliance in chronic obstructive hydrocephalus is also decreased. The hydrodynamics of chronic obstructive hydrocephalus is similar to that of chronic communicating hydrocephalus, despite their different origins. Common to both types of chronic hydrocephalus is the breakdown of the windkessel mechanism with significantly increased pulsations of the brain capillaries. A physiological link from the acute phase to the chronic phase of obstructive hydrocephalus is established, that is a vascular adaptation causing a favourable decrease in the CSF pressure decreasing venous congestion and an unfavourable compression of the intracranial capacitance vessels.

At least three different treatment options of communicating hydrocephalus are available, shunting, third ventriculostomy and posterior fossa decompression. Shunting will probably remain the most effective and used treatment in the near future. However, successful shunting is more drastic and not as physiological as the other treatments. It is based on a slight and in some cases even extensive, over-drainage of CSF and is suffering under a high complication rate. Posterior fossa decompression has the advantage of being a causative treatment of communicating hydrocephalus caused by obstructions at the craniocervical junction. Shunting and third ventriculostomy are symptomatic treatments also aimed at increasing the intracranial compliance. The indication for performing third ventriculostomy, as a first line treatment of communicating hydrocephalus, must be further tested and remains to be determined. Third ventriculostomy may in several instances replace a shunt and make the patient shunt independent.

An understanding of the hydrodynamics in hydrocephalus increases the diagnostic accuracy of radiological examinations. This is important, since the diagnosis and treatment of hydrocephalus is based on a combination of the radiological findings and the clinical picture. Theories concerning the capillary absorption of CSF including the hydrodynamic view on hydrocephalus presented here will continue to evolve. It is clear that increased knowledge of the pathophysiology behind hydrocephalus will improve therapy. Future therapy of chronic hydrocephalus will aim at increasing intracranial compliance and reducing pulse pressure rather than draining CSF. To adhere to the CSF bulk flow theory without considering hydrodynamic mechanisms cannot be considered state of the art any longer.

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